

The Importance of Middle Cerebral Artery Stenosis In Patients With A Lacunar Infarction In The Carotid Artery Territory

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Background : It is well known that a lacunar infarction may develop by an atherosclerosis of the large intracranial arteries at the site of the perforating arteries. However, their frequency, clinical, and radiological findings have rarely been described. **Methods :** Carotid angiographies were carried out in 26 Korean patients with lacunar infarctions in the carotid arterial territory. They all had classical lacunar symptoms with a computed tomogram (CT) or a magnetic resonance image (MRI) evidence of a small infarction. A transesophageal echocardiography (TEE) was carried out in fifteen patients when either an angiography result was normal, the potential source of cardioembolism was suggested, or no other atherothrombotic causes of stroke were found. Tc-99m single positron emission computed tomograms (SPECT) were performed in nine patients. We divided patients into two groups; one for patients with MCA occlusive lesion, and another for those without it. The clinical and radiological features were compared between the two groups. **Results :** Twenty patients (77%) demonstrated abnormal angiographic findings. Fourteen of them showed atherosclerotic changes in the proximal MCA at the site of the orifice of the lenticulostriatal arteries, while another three showed stenosis in the intracranial portion of the internal cerebral artery (ICA), and the other three in the extracranial ICA. Among six patients with normal angiograms, a TEE demonstrated embolic sources of embolism in two patients. The temporal profile and findings of MRI and SPECT in patients with MCA stenosis differed from those with ICA stenosis or normal angiograms. Unstable temporal profiles exclusively occurred in patients with MCA stenosis. The most patients with conglomerate aggregations of the lacune in a MRI showed MCA lesions. The SPECT findings were even more characteristic in that patients with MCA lesions showed relatively large areas of decreased perfusion. **Conclusions :** The atherosclerotic diseases at the orifice of the lenticulostriatal arteries were the most common causes of lacunar infarctions in the carotid artery territory. They were clearly different from those without MCA occlusive lesions in terms of preceding transient ischemic attacks, unstable temporal profiles, unilateral multiple lacunes with conglomerate MRI findings, and widespread perfusion defects in SPECT.

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Key Words : Lacunar Infarction, Angiography, Atherosclerosis, Embolism, Cerebrovascular Disease

(lacunar infarction) 가 , lacunar infarction
가

Lacunar infarction 가
(small vessel disease) 가
(ICA)
lacunar infarction^{1,2}
(MCA) trunk (M1 portion)

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lenticulostriate artery (parent artery) lacunar infarction^{3,4,5}

가 ,^{3,5}

striatocapsular infarction MCA^{3,5}

가 .

lacunar infarction

MCA

, MCA

(MRI),

(SPECT)

1994 10 , 1996 7

pure motor (PMH), sensorimotor stroke (SMS), ataxic hemiparesis (AH) dysarthria-clumsy hand syndrome (DCH) lacunar syndrome

15mm small deep infarction lacunar infarction

lacunar infarction (basal ganglia), (corona radiata), centrum semiovale

(car-

dioembolism)

(transesophageal echocardiography(TEE))

160/90mmHg

(%)

가 ,

Brain

MRI 26 23

Nelson 1 ml

⁶ Tc-99m SPECT 9

, 21 (tran-

scranial Doppler study(TCD))

가

1

NIH

scale 2

가 2 “improve”,

“stable”, 가

“worsening”, 가 가

가 “fluctuation” .

lenticulostriate artery가

MCA trunk (M1 portion) mural athero-

ma가 가

MCA (irregularity) (steno-

sis) ,

MCA lacunar infarction

1.

431 (1)

가 , 122 lacunar infarc-

tion 122 73 anterior

circulation . 165

atherothrombotic stroke, 74 cardioembolic

stroke, 10 other determined,

가 60 .

431 118

46

11 19

35 가

lacunar infarction 26 가

carotid artery lacunar infarction

17 9

55.8 (29-80) .

Table 1

2. Lacunar infarction

14 MCA lenticulostriatal arteries

가 , 2 가

(<50%), 5 가 (50-74%), 4 가

(>75%) , 3

(Table 2 Fig. 1).

12 MCA lacunar infarction

가 7

5

(Table 2). TEE 15

3 Patent foramen ovale

with aortic arch atheroma, aortic arch athero-

ma, cardiac echogenic mass가

Table 2

3. MCA (Table 3)

Table 1. Patients with striatocapsular territory lacunar infarction included in this study

case	sex/ age	risk factors	syndrome	TIA	temporal	angiography profile	MRI	site	SPECT
1	F/65	cardiac mass	PMH	-	stable	normal	bilateral multiple	BG	not done
2	M/68	HiBP, DM	DCH	-	improving	M1 stenosis	unilateral single	BG	not done
3	F/56	HiBP	PMH	-	worsening	M1 stenosis	bilateral multiple	CS,BG	global subCx, Cx
4	F/46	HiBP	SMS	-	improving	normal	bilateral multiple	CS	normal
5	M/80	HiBP,smoking	DCH	-	fluctuating	M1 stenosis	unilateral multiple	CR	not done
6	M/66	HiBP	AH	-	improving	normal	not done	CR	not done
7	F/29	HiBP, DM	PMH	+	fluctuating	M1 stenosis	unilateral multiple	BG	not done
8	M/47	DM, smoking	SMS	-	stable	normal	not done	-	not done
9	M/40	HiBP,smoking	PMH	-	stable	M1 stenosis ICA(I)	unilateral single	BG	not done
10	M/53	none	PMH	-	stable	irregularity M1, stenosis	bilateral multiple	BG,CS	subCx, adjacent Cx
11	M/68	HiBP	DCH	+	improving	ICA(I,E) stenosis	bilateral multiple	BG,CS	not done
12	M/45	HiBP	PMH	-	worsening	M1 irregularity	unilateral multiple	BG	global subCx, Cx
13	M/59	DM,smoking	AH	-	improving	ICA(I) stenosis	bilateral multiple	BG	subCx, adjacent Cx
14	M/56	HiBP	PMH	-	stable	ICA(I) stenosis	bilateral single	CR	not done
15	F/68	DM	DCH	-	stable	ICA(E) stenosis	unilateral single	BG	not done
16	F/64	HiBP, AAA	PMH	-	stable	ICA(E) stenosis	unilateral multiple	CS	not done
17	M/66	AAA, PFO	SMS	-	improving	normal	bilateral multiple	BG,CR	not done
18	F/56	HiBP	PMH	-	stable	normal	unilateral single	CR	small subCx
19	M/62	HiBP,smoking	SMS	-	worsening	ICA(E) stenosis	unilateral single	CS	not done
20	M/49	HiBP	SMS	+	improving	M1 stenosis	unilateral single	BG	not done
21	M/30	none	SMS	+	improving	M1 stenosis	unilateral single	BG	not done
22	M/56	HiBP,smoking	PMH	-	stable	M1 irregularity	unilateral multiple	BG	subCx, adjacent Cx
23	M/61	HiBP	SMS	-	improving	M1 irregularity	bilateral multiple	BG	subCx, adjacent Cx
24	F/68	HiBP, RHD	SMS	-	improving	M1 stenosis	unilateral multiple	CR	global hemispheric
25	F/32	HiBP, DM	PMH	+	fluctuating	M1 stenosis	not done	-	not done
26	M/63	DM, smoking	PMH	-	stable	M1 stenosis	unilateral multiple	BG,CR	not done

TIA; transient ischemic attack, HiBP; hypertension, DM; diabetes mellitus, AAA; aortic arch atheroma, PFO; patent foramen ovale, RHD; rheumatic heart disease

PMH; pure motor hemiparesis, SMS; sensorimotor stroke, AH; ataxic hemiparesis, DCH; dysarthria-clumsy hand syndrome.

M1; proximal portion of middle cerebral artery, ICA(E); extracranial portion of internal cerebral artery, ICA(I); intracranial portion of internal cerebral artery,

BG; basal ganglia, CR; corona radiata, CS; centrum semiovale

subCx; subcortical area Cx; cortical area

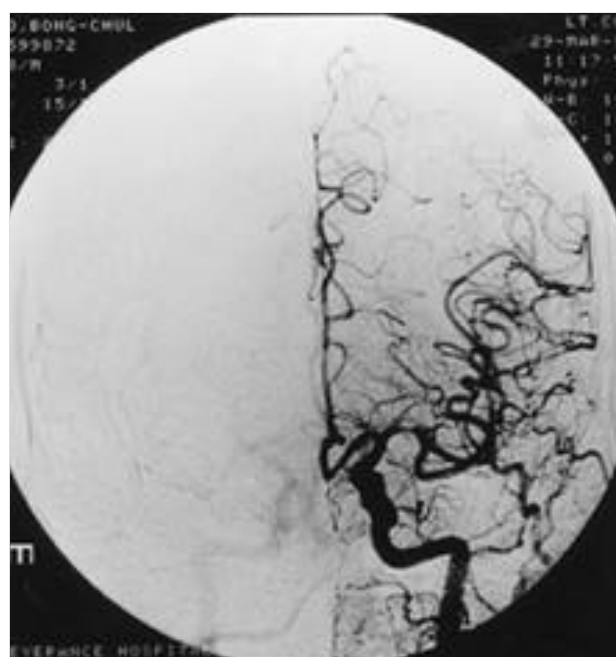


Figure 1. Cerebral angiography: Severe stenosis of middle cerebral artery at the site of origin of the lenticulostriate artery

Table 2. Angiographic classification of 26 patients of lacunar infarction.

Angiographic finding *	Cardioembolic source		Total
	(+)	(-)	
MCA proximal stenosis	0	13	13
Severe irregularity only			3 (23%)
Stenosis < 50 %			1 (8%)
50-74 %			5 (38%)
> 75 %			4 (31%)
ICA lesion	1	5	6
extracranial	1	2	3
intracranial	0	3	3
MCA proximal and ICA lesion	0	1	1
Normal	2	4	6
Total	3	23	26

* stenosis of symptomatic side

Table 3. Clinical features in each type of lacunar infarctions.

	No MCA lesion	MCA stenosis
Patients(%)	12 (46 %)	14 (54 %)
Age < 45	0	5
46-60	6	3
> 60	6	6
Sex man	7	10
women	5	4
Risk factors		
hypertension	6	12
diabetes mellitus	3	4
smoking	3	4
Temporal profile(%) *		
improving	4 (33 %)	6 (43 %)
stable	7 (58 %)	3 (21 %)
fluctuating	0 (0 %)	3 (21 %)
worsening	1 (8 %)	2 (14 %)
Previous TIA episodes	0	5
Lacunar syndrome type(%)		
pure motor hemiparesis(PMH)	5 (42 %)	7 (50 %)
sensory motor stroke(SMS)	4 (33 %)	4 (29 %)
dysarthria clumsy hand syndrome	1 (8 %)	3 (21 %)
ataxic hemiparesis	2 (17 %)	0 (0 %)

* Neurologic status was evaluated upon admission and after 1, 3, and 7 days of admission.

Clinical course was defined by NIH score as follows :

improve, lowered 2 points or more ;

stable, lowered less than 2 points ;

fluctuating, decrease after episodic increase, or vice versa ;

worsening, increase after admission.

MCA : middle cerebral artery

TIA : transient ischemic attack

Table 4. Radiological features in each type of lacunar infarction.

	No MCA lesion	MCA stenosis
(1) MRI findings	n = 10	n = 13
Site of infarction *		
BG or CR	6 (60 %)	11 (85 %)
BG	3	8
CR	2	2
BG and CR	1	1
CS	3 (30 %)	0 (0 %)
mixed	1 (10 %)	2 (15 %)
Volume of infarction **		
1 ml	8	9
< 1 ml		4
Laterality of lacunae (%)		
unilateral single	3 (30 %)	4 (31 %)
bilateral single or multiple	6 (60 %)	3 (23 %)
unilateral multiple(2)	1 (10 %)	6 (46 %)
leukoariosis	4	3
(2) Low perfusion area in SPECT	n = 4	n = 5
normal or small subcortical	2	0
focal in subcortex and adjacent cortex	2	2
global in subcortex and cortex ***	0	3

* BG : basal ganglia, CR : corona radiata, CS : centrum semiovale,
mixed : BG or CR and CS

** by the method of Nelson et al 6

*** area of decreased perfusion in SPECT exceeded the area of signal changes in MRI



Figure 2. MRI findings: Unilateral multiple lacunae with conglomeratory aggregation at the left hemisphere in the case of a middle cerebral artery occlusive lesion

MCA
lacunar syndrome
(fluctuation
worsening) 가 MCA
(5), 1
ICA
5
MCA
4. MCA (Table 4)
MRI 23 Lacunar infarc-
tion MCA basal ganglia
corona radiata 가
(85%), centrum semiovale
MCA basal ganglia
corona radiata centrum semiovale
ICA 가
(67%).
Lacune

MCA 가 8 (62%) MCA
ICA 2 MRI
lacunae가 conglomeratory aggregation
(Fig. 2) CT multiple lacune
15mm

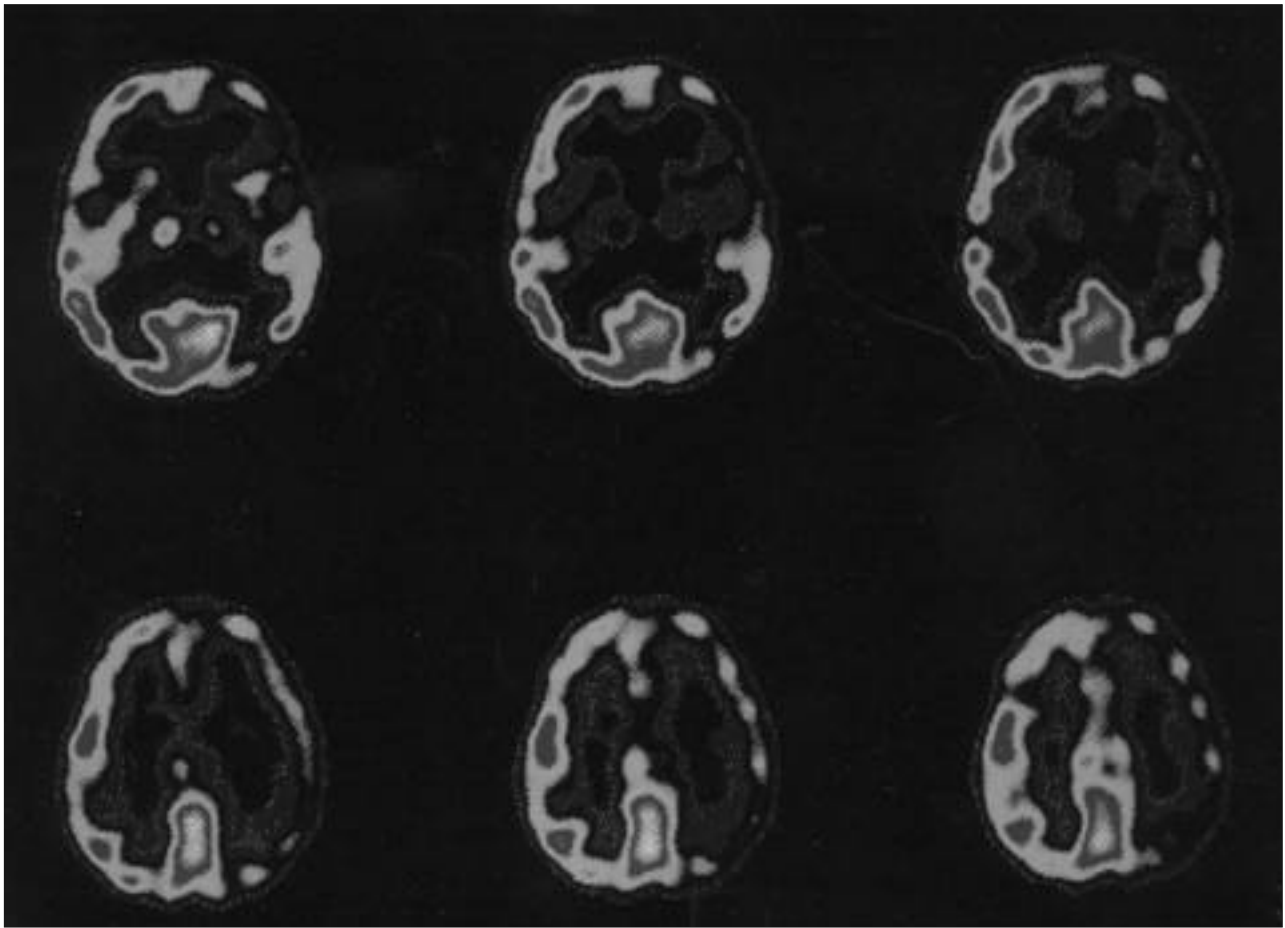


Figure 3. SPECT findings: Compared to MRI findings shows marked decreased perfusion in the case of lacunar syndrome with middle cerebral artery occlusive lesion

7
(multiple lacune)가 ,
7 6 MCA lacune MCA 가 .7 spasm turbulent flow
ICA MCA lacunar infarction
leukoariosis , nar infarction , lacu-
(71.4%). lostriatal branch lenticu-
SPECT 9 4 .1 lacunar infarc-
MCA 5 tion (carotid
가 MRI bifurcation)
SPECT MCA 5 .1,8
(Fig. 3), ICA 가 cortical infarction striatocapsular
infarction ,9,10
22% .1 ICA
lacunar infarction 가 ,3 lacunar infarction
가 , 가
lacunar infarction MCA branch lacunar infar-
ction
가 , 가 MCA
penetrating artery medullary artery MCA

30% lacunar infarction³

reversible ischemic neurologic deficit (RIND)가 capsular lacunar infarction 16

11 MCA M1, 4 ICA

infarction 4% capsular¹¹

lacunar infarction MCA, 26 capsular lacunar infarction 14 MCA stem, 7

ICA lacunar infarction MCA

Lacunar infarction (clue)가⁵

infarction lacunar stroke

non-lacunar stroke가, ¹⁴

MCA lacunar infarction, lacunar

infarction가¹²

(/ ,)¹³ lacunar infarction

. Thajeb capsular lacunar syndrome, MCA ICA⁵ Bogousslavsky MCA lacunar infarction 11.7%

³ MCA

가 .

Lacunar infarction lacunar infarction가

가 .

가

. , MRI

가

lacunar infarction

leukoariosis lacunar infarction underlying small vessel vasculopathy가¹⁵

MCA MRI

MCA lacunar infarction가

lacune conglomeratory aggregation ICA

microembolism lacunar infarction MRI, MRI

lacune TEE가

leukoariosis lacunae, SPECT TCD lacunar infarction

striatocapsular infarction SPECT가 lacunar infarction가¹⁶

MCA main trunk striatocapsular infarction lacunar infarction

lacunar infarction

MCA SPECT

MRI가

lacunar infarction SPECT MCA

capsular lacunar infarction, MCA lacunar infarction

MCA

MRI, SPECT lacunar infarction

TCD

TCD SPECT lacunar infarction

lacunar infarction가

REFERENCES

1. Kappelle LJ, Koudstaal PJ, Gijn J, Ramos LMP, Keunen JEE. Carotid angiopathy in patients with lacunar infarction; A prospective study. *Stroke* 1988;19:1093-1096.
2. Horowitz DR, Tuhim S, Weinberger JM, Rudolph SH. Mechanism in lacunar infarction. *Stroke* 1992;23:325-327.
3. Bogousslavsky J, Barnett HJM, Fox AJ, Hachinski VC, Taylor W. Atherosclerotic disease of the middle cerebral artery. *Stroke* 1986;17(6):1112-1120.
4. Fisher CM. Lacunar infarcts : A review. *Cerebrovasc Dis* 1991;1:311-320.
5. Thajeb P. Large vessel disease in Chinese patients with capsular infarcts and prior ipsilateral transient ischemia. *Neuroradiology* 1993;35(3):190-195.
6. Nelson RF, Pullicino P, Kendall BE, Marshall J. Computed tomography in patients with lacunar syndromes. *Stroke* 1980;11:256-261.
7. Pullicino PM. Pathogenesis of lacunar infarcts and small deep infarcts. In: Pullicino PM, Caplan LR, Hommel M, eds. *Advances in Neurology* Vol 62. Raven press, New York, NY, 1993;125-129.
8. Ghika J, Bogousslavsky J, Regli F. Infarcts in the territory of the deep perforators form the carotid system. *Neurology* 1989;39:507-512.
9. Boiten J, Lodder J. Larger striatocapsular infarcts: Clinical presentation and pathogenesis in comparison with lacunar and cortical infarcts. *Acta Neurologica Scand* 1992;86(3):298-303.
10. Lindgren A, Roijer A, Norrving B, Wallin L, Eskilsson J, Johansson BB. Carotid artery and heart disease in subtypes of cerebral infarction. *Stroke* 1994;25(12):2356-2362.
11. Tei H, Uchiyama S, Maruyama S. Capsular infarcts: Location, size and etiology of pure motor hemiparesis, sensorimotor stroke and ataxic hemiparesis. *Acta Neurologica Scand* 1993;88(4):264-268.
12. Clavier I, Hommel M, Besson G, Noelle B, Perret JE. Long-term prognosis of symptomatic lacunar infarcts: A hospital-based study. *Stroke* 1994;25(10):2005-2009.
13. Fisher CM. Bilateral occlusion of basilar artery branches. *J Neurol Neurosurg Psychiatry* 1977;40:1182-1189.
14. Boiten J, Lodder J. Lacunar infarcts. Pathogenesis and validity of clinical syndromes. *Stroke* 1991;22(11):1374-1378.
15. Boiten J, Lodder J, Kessels F. Two clinically distinct lacunar infarct entities?; A hypothesis. *Stroke* 1993;24(5):652-656.
16. Isaka Y, Imaizumi M, Ashida K, Nakayama H, Iiji O, Itoi Y, Furukawa T. Relationship between extent of brain hypoperfused area and functional outcome in patients with a small subcortical infarction: evaluation with X-ray CT, 123I-IMP cerebral perfusion SPECT and cerebral angiography. *Kaku Igaku-Japanese Journal of Nuclear Medicine* 1992;29(1):1-8.